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EDITORIAL

It is a sadder and wiser pen that we lift for the last time in the editorial columns of this journal. For with the going to press of this issue, the time of the year and the proximity of the examinations is brought forcibly upon us.

And while the thought of examinations is still upon us, let us be placed on record as saying that we will pass them successfully no matter what is in store for us; from the sixth year down, let us not fail. The usual plaintive wail of the final year man, like a bleating lamb on a sodden moor has been, "you have pushed us through five years, will you not push us once more?" We openly and vigorously decry that attitude as being the mark of an acamedic cad. Let us rather hope that our knowledge is sufficient so that we may be allowed, honestly, to go forth and practice medicine. If we be tried in the balance and found wanting, let us be adjudged so for the sake of those whom we will serve.

It has been said of the surgeon that he must have the strength of a giant, the courage of a lion, the eye of an eagle, and the gentleness of a woman's hand. That should be true of all medical men. So let us go forth, those of us who are in the final year, with high courage, for it will be tried, with

great strength, for it will be sapped, with a keen eye, for it will be dimmed, and with a gentleness in our dealings with suffering humanity that never becomes roughened. That is our trust. We accept it, and carry on.

PATHOLOGICAL CONFERENCES.

There is one institution in the sixth year that merits applause, namely the Pathological Conferences held every Thursday afternoon in the Banting Institute. For the enlightening of the uninitiated, these are symposia on various prominent topics in which the combined knowledge of Anatomists, Physiologists, Pathologists and Clinicians is brought to bear upon one disease entity. Such symposia do much to correlate the basic subjects of Medicine and Surgery with the clinical aspects of disease. But further, points and problems appear which even our peers are not agreed upon. That is, the study of medicine is made dynamic rather than static and the attitude that medicine is not a living subject receives a rude jolt; the result is that hope is roused in the student's mind that some day he will be able to aid in the solution of some of these problems. Lastly, the fact that the students can and are encouraged to take part in the discussion is to be highly commended. There are those with a thought process so poverty stricken that any divergence from spoon feeding is viewed with intense consternation. Their bleating for milk and arrowroot should be ignored, it is time they were weaned.

Great appreciation should be extended to those who are active in arranging and conducting these conferences. May they continue to receive the popular approval which they so well deserve.

Before taking our final leave we would like to point out some of the accomplishments of the Journal and to give thanks to those who made such advances possible. To those of the Staff who have contributed to the Journal this year, we express our grateful thanks. They have contributed authoritative articles, and by so doing we may hope to gain somewhat in kudos in the eyes of other medical publications. Let us hope that they will continue to favour the Journal thus, so that, though we wish to remain a student publication, yet we wish to be known as a good one.

To the managing editor, Mr. Hugh Bright and his assistant and the year representatives we are especially indebted for the excellent organization of the circulation of the Journal, and also for his efforts which have been solely responsible for the financial success of this past year. To the assistant editor and the associate editors we are grateful for their aid in proof-reading, obtaining copy, and their general interest and helpfulness. It has been the co-operation of these members of the Journal staff that has enabled the Journal to increase its circulation over previous years, and has made the position of editor a very pleasant and enjoyable one indeed.

Lastly we would like to extend our thanks to those who made the Journal possible, our subscribers and our advertisers, who by their loyal support have made our task an easy and a pleasant one.

The Journal will be in the very capable hands of Mr. O. M. Solandt during the next year so we take our final leave knowing that the Journal will achieve those ideals of which we have fallen short.

THE ESTIMATION OF BLOOD IODINE

By DAVID L. SELBY

Department of Pathological Chemistry,
University of Toronto.

Iodine was discovered by Courtois in the year 1812, who isolated it from kelp or burnt sea weed. It does not occur naturally as the free element but always combined, usually as the iodide or iodate of sodium or potassium and is widely though sparingly distributed. It occurs in sea water and therefore in most sea plants, and is present in the salt petre deposits of Chile as an impurity from which it can be isolated.

Iodine is widely used in medicine, the most common form being the iodide of potassium. For the past 100 years the biochemical study of goitre has centered around this element. However, long before this, iodine was used empirically in the treatment of goitre. Even in 1200 A.D., Roger Von Solerno administered sponges and seaweeds to help his patients who had swellings in the neck. However, it was not until 1829 that Coindet showed that the active agent which gave such substances their therapeutic value was iodine. Prout in England used potassium iodide in the treatment of goitre in 1816. In 1819 this treatment was adopted in St. Thomas' Hospital.

Chatin advanced the theory that since iodine distribution was poor in districts known for endemic goitre, the administration of iodine might act prophylactically. He was ridiculed and his ideas dismissed only to be revived at a much later date.

Baumann appointed by the Swiss Commission on goitre, demonstrated in 1895 that iodine is a normal constuitnent of thyroid gland and that it is present in combination with a globulin. He also demonstrated that hyperplastic glands were low in iodine content.

In 1897 Oswald showed that in man and in calves the iodine content of the thyroid gland varied with the amount of colloid in the gland. Marine et al confirmed this. Thus he found that the exophthalmic gland had the lowest content of iodine, the normal gland the highest amount and the colloid gland slightly less than normal. Gley and Bourcet in 1900 were the first to show that iodine was a constant and normal constituent of the blood. They managed to isolate it from a very large quantity using 200 litres.

Marine however, is responsible for most of the modern work on the use of iodine in phophylaxis and treatment of goitre. In 1917 Marine reported the results of an investigation carried out on the school children of Akron, Ohio, of the prophylactic and therapeutic effect of iodine in adolescent goitre.

Most of the iodine in biological material is in organic combination. In the sponge and coral the compound is diiodotyrosine. In 1916 Kendall isolated thyroxin from the thyroid gland. Harington however, discovered its structure and was the first to synthesize it. Thyroxin itself is found only in traces in the gland, the bulk of it being in protein combination, usually named iodothyroglobulin. Diiodotyrosine has been isolated from hog thyroid.

Extensive studies of iodine in blood and thyroid tissue have been made by a vast army of investigators. The maximum content of iodine in the thyroid gland is about 25 mgms. In comparison to this, blood contains a minute amount normally, ranging from 10-15 gamma (Y) per 100 cc. (.000010-.000015 gm.). Approximately 50% of the total iodine of the body is found in the thyroid gland, the remainder being distributed throughout the blood and body tissues.

Curtis, Davis and Phillips found that in Chicago the average blood iodine value of 12 Y %. This level was slightly lower in winter averaging 10 Y %. They give the normal range as being from 8.5 Y to 16.2 Y %.

Turner in Cleveland studied the iodine content of the bloods of individuals in hospital and outpatient clinics. Of the 101 cases studied, 38 were normal averaging 12 Y % ; 32 were subnormal averaging 7 Y % and 31 were above normal averaging 60 Y %. Further all normal and non pathological cases were within normal limits averaging 11 Y %.

McCullagh and Picha devised a method by which they estimated total iodine in about 3 hours time and used it in the study of thyroid diseases. They found the normal range in winter to be between 6 Y and 9 Y %.

Perkin, Brown and Lang using a method devised by Perkin, in which the error is less than 10% found that in normal individuals the blood iodine content lay between 2.4 Y and 18.5 Y %. Under basal resting conditions the normal range was 2.2 Y to 6.4 Y %. In hyperthyroidism they found a range from 7.2 Y to 141 Y %.

The work of Holst, of Lunde and of Veil and Sturm, is particularly interesting. They found that the total iodine of the blood was made up of two fractions, one soluble in ethyl alcohol which is supposedly inorganic iodine and the other insoluble in ethyl alcohol which apparently is organic iodine. Normally they found that the insoluble fraction made up approximately 2/3 of the total, a fact not accurately confirmed, by other investigators. To these authors the organic or alcohol insoluble iodine was the more interesting as the amount of this fraction appeared to be an excellent measure of thyroid activity. They stated that it was low in individuals with hypothyroidism and high in cases of hyperthyroidism.

From this fact they came to the conclusion that estimations of total iodine were of little value as the total blood iodine level rose and fell with the intake and excretion of inorganic iodides and iodates but that estimations of the alcohol insoluble fraction as mentioned above was a measure of the substance responsible for the toxic symptoms of hyperthyroidism. Normally they found that the alcohol insoluble fraction was constant,giving a range of 1-4Y% and that the alcohol soluble fraction varied from 7-12 Y %. Following the administration of inorganic iodine in normals the alcohol soluble fraction rose sharply while the alcohol insoluble fraction remained constant.

In a series of cases of hyperthyroidism the alcohol insoluble values were all above normal as were the basal metabolic rates. When iodine was administered by mouth, the basal metabolic rate fell as Plummer had shown previously. Co-incident with the fall in basal metabolic rate they found the alcohol insoluble fraction fell even though inorganic iodine was being administered therapeutically. Following thyroidectomy performed at the proper time both the basal metabolic rate and the alcohol insoluble iodine fraction remained within normal limits.

Dodds, Lawson and Robertson confirmed the fact that the blood iodine was divisible into alcohol soluble and alcohol insoluble fractions. They also noted that in hyperthyroidism the alcohol insoluble fraction was generally high and fell off after administration of iodine by mouth. They did not however, find that the basal metabolic rate or the symptoms of toxicity in the thyroid bore any relationship to the alcohol insoluble fraction. They showed that often, high toxicity and high basal metabolic rates were present along with low alcohol insoluble iodine values and vice versa. They concluded that the solution of the problem of the causes of toxicity in hyperthyroidism was not to be found in the alcohol insoluble fraction of the blood iodine.

In patients with diffuse hyperplastic goitre the blood iodine is consistently elevated. It falls slightly on rest in

bed. After "adequate" thyroidectomy the blood iodine falls to normal but after "inadequate" thyroidectomy it remains elevated.

In toxic adenoma as in exophthalmic goitre the blood iodine is raised but usually not to the same height as in the diffuse hyperplastic disease. Following "adequate" thyroidectomy the blood iodine values fall to normal as in diffuse goitre. In non-toxic goitre the blood iodine is usually within the normal range.

The blood iodine becomes elevated in patients treated with large amounts of thyroid extract. It may become as high as is found in exophthalmic goitre. The blood iodine is not markedly lowered in cases of hypothyroidism. Indeed it usually is found to fall in the low range of normal. This observation has been made in cases of cretinism, myxoedema, and in colloid goitre accompanied by low basal metabolic rate.

Physiologically, the blood is found to vary considerably. Exercise raises the blood iodine sharply but there is a rapid return to normal, and even below normal, so that 30 minutes after the completion of violent exercise the blood iodine is often subnormal. Iodine in the blood is raised due to trauma as shown in surgical operations. The iodine seems to be raised in proportion to the amount of the trauma often being increased several hundred percent. Varying effects were observed by Leitch following the use of anaesthetics, the blood iodine usually being increased but occasionally decreased. That the iodine comes from the thyroid gland seems logical as no iodine was used in the operating room in the preparation of these patients. Leitch found that in goats the blood iodine value varied with the sexual cycle and in the human female the value is elevated during the first stages of menstruation and during pregnancy and is still higher in pregnancy if the individual is taking iodine in some form.

McCullagh and Picha confirmed the fact that in women there is a sharp increase in blood iodine during the first few days of menstruation. During the mid-menstrual period the normal level is the same as in men. Other glands of internal secretion besides the ovary have an effect on blood iodine. Closs, Loeb and MacKay showed that pituitary extract influences blood iodine. They injected an acid extract of anterior pituitary and found that they could increase the basal metabolic rate, induce changes in the thyroid gland suggestive of Grave's disease, and raise the blood iodine. They further found that when blood iodine was raised total thyroid iodine was lowered.

Turner in Cleveland investigated the blood iodine in patients from the out-patient clinic with the following results. He found that in 67 diabetics, 20 were normal (average 11 Y %); 28 were subnormal (average 7 Y %); 19 were above normal (average 53 Y %). Of 15 hyperthyroid cases 5 were normal (11.5 Y %) and 10 were above normal (87 Y %). Of 4 hypothyroid cases all were normal (10 Y %). Of 15 miscellaneous cases 9 were normal (12 Y %); 4 were subnormal (6 Y %), and 2 were above normal (67 Y %).

McCullagh and Picha feel that the blood iodine is consistently raised in hyperthyroidism although there is no relationship between the height of the blood iodine and the basal metabolic rate. They quote experiences in which the basal rate was high but the blood iodine was normal. These patients were adequately treated in hospital without operation. In other cases the reverse was found, i.e., normal basal metabolic rate and high blood iodine, with the results that improvement did not follow till operation had been performed.

Gordon and Graham report clinical evidence of hyperthyroidism with normal basal metabolic rate. These patients were remarkably improved by operation. Blood iodine studies were not reported in this paper. These cases usually occurred in the group of nodular goitres. It is worth noting that the systolic blood pressure and pulse rate were higher in the group of normal basal metabolic rate—hyperthyroid

cases than in the strictly normal patients.

Curtis and Cole confirm many of the foregoing facts and show that blood iodine after total thyroidectomy remains at about 1/3 the normal level. This level is presumably maintained by food and water intake. Curtis believes that blood iodine is comparable in diagnostic significance in cases of hyperthyroidism to studies of blood sugar in diabetes or of blood calcium in parathyroid disease. Concerning this statement it is interesting to note that Perkin, Brown and Lang devised an iodine tolerance test described briefly as follows: A "fasting" sample of blood is drawn, followed by the oral administration of 10 minims of Lugol's iodine in milk. Blood samples are taken again at ½, 1, 1½, 2½ and 7 hour intervals. Urine iodine estimations are also done, the diet being free of foods high in iodine content. They found that indididuals with hyperthyroidism gave lower iodine tolerance curves than normals. They do not attempt to explain the significance of these results. One would think that iodine was rapidly removed from the circulation probably by the thyroid gland. Indeed the lower urinary excretion in hyperthyroid patients would confirm this. Plummer and others have shown the value of iodine in treatment of hyperthyroid cases and as it is known that the hyperthyroid glands contain less iodine than normal glands. the removal of iodine from the blood, causing a lower tolerance curve, would seem logical.

Conclusions.

1. Normally the blood iodine is maintained at a fairly constant level varying somewhat in different regions and with different methods of estimation.

2. The blood iodine is made up of two fractions, organic and inorganic or alcohol insoluble and alcohol soluble.

3. In hyperthyroidism the blood iodine is fairly consistently elevated. It seems questionable whether or not the alcohol insoluble fraction is the important factor.

4. In hypothyroidism low normal results are obtained. After complete thyroidectomy the blood iodine falls to about 1/3 of normal.

5. In women the menstrual cycle raises the blood iodine. In the midmenstrual period the normal values are the same for women as for men.

6. The pituitary gland as well as the thyroid and ovarian glands apparently has an effect on blood iodine.

7. Violent exercise and trauma raise the blood iodine.

8. When the iodine content of the blood is raised, that of the thyroid gland is diminished and vice versa. The thyroid gland normally contains 50% of the total iodine.

9. In the diagnosis of certain cases of hyperthyroidism with normal metabolic rate the estimation of blood iodine would appear to be helpful.

10. The possibility of the use of an iodine tolerance test as an aid in the diagnosis of hyperthyroidism has been discussed.

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THE NURSING OF SURGICAL CASES SUFFERING FROM THYROID INTOXICATION

A GENERAL ROUTINE OF TREATMENT ADOPTED IN THE SURGICAL WARDS AT THE TORONTO GENERAL HOSPITAL.*

The aim of the pre-operative treatment of patients suffering from thyroid intoxication is to secure physical and mental rest. With this end in view patients are admitted where possible, to small or single bed wards, where quiet will be assured. A few whose condition justifies it are allowed bath-room privileges, but the majority are kept at absolute rest in bed. Since these patients often do not sleep until late at night, every effort compatible with hospital routine is made to leave them undisturbed when sleeping in the morning. Patients suffering from thyroid intoxication on this Service are given Luminal grains 1 at bedtime to insure rest. If this is inadequate the dose is increased to Luminal grains 1 at bedtime and grains ½ three times a day. Visitors are restricted to the minimum, to those, in fact, whom it would not distress the patient to see. Exciting or disturbing literature is definitely banned. With a quiet room, comfortable bed, sufficient sedative, restricted visitors and activities, an average patient has a good chance of rest. A controlling factor, however, in the care of such patients is the co-operation of the patients themselves. It is a wise course early in a patient's hospital career to spend sufficient time explaining the plan of treatment, the way in which they may co-operate and why it is essential. A patient suffering thyroid intoxication is by no means an easy one to nurse. She is an apprehensive and irritable individual, given to tears and terror, convinced that her operation will be sprung upon her without warning, uncertain of the disposition of everyone around her, and most of all uncertain of herself. Nursing is undoubtedly a most important factor in the re-establishment of the patient's self-control and confidence. For this reason we try to arrange for the same nurses remaining in charge of the nursing care of these patients throughout the stay in hospital so that the disturbance of change and re-adjustments is reduced to a minimum. For the same reason, such patients on this Service do not undergo the test for Basal Metabolic Rate without an explanation the night before. On a large and busy ward this is sometimes easy to overlook. To these patients the unknown is full of dire possibilities and a B.M.R. test with its curious paraphernalia is just added apprehension to a patient whose resistance to disturbance is already low. Carried out chemically it is a breathing test to determine the rate at which the body as a whole consumes oxygen and produces carbon-dioxide. It is measured when the body is at rest, when the temperature is normal, when physical and mental activities are at a minimum, and when digestive processes and their stimulating effects are at the lowest level. It is necessary to know the patient's height and weight for these tests, which is usually carried out in the morning within a few days of admission. The patient is left undisturbed that morning until it is due, no food being allowed and the toilet reduced to a minimum.

Thyroid patients usually have a good appetite and are willing to drink what is required of them. They should have a simple diet of high caloric value,

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* This routine is not absolute, varying from ward to ward, and from surgeon to surgeon, and even from case to case, but as a guide to students should prove invaluable.—Ed.

suppemented by milk, cocoa, etc., between meals. The daily fluid intake should be at least one hundred ounces and in this we include one quart of glucose drink which is made as follows: One pound of syrup glucose, the juice of one orange and two lemons with their pulp cut in fine pieces, water to make one quart. Mix and bring to a boil. Let boil for five minutes. Make the volume up to one quart and serve ice cold.

So far no mention has been made of Lugol's Iodine, but with intent. Iodine is, of course, the most valuable ally in the pre-operative care of the thyroid patient. Iodine alone cannot produce the really amazing results that occur when it is used in combination with other measures, i.e., physical and mental rest and adequate nourishment and fluids. Our medical routine, started within a few days of the patient's admission, is as follows: Lugol's iodine minims 10, three times a day. With the administration of Lugol's iodine for the first two days only we give "Digifolium minims 20 three times a day, and thereafter minims 10 daily. Lugol's iodine, being an unpalatable medicine, is given in milk or grape juice. It is interesting to watch the gradual drop of a patient's pulse curve after the administration of iodine. The majority of cases will strike their lowest level within fourteen days and that is the point at which we operate, before the curve starts to swing up again on its new cycle uncontrolled by iodine. It is no less interesting to watch the change in the patient's reactions. The Doctor's morning visit, which was once greeted by an air of startled agitation and a rapidly beating pulse, is now regarded not as a matter of disturbance, but as a pleasant incident of the day. The patient's bed, which was at first in a constant state of disarranged unheaval, gradually takes on a seemly orderliness. The real apprehension of operation changes to a rational unperturbed interest in when it may take place. From observation of these cases, we have come to the conclusion that the nursing care in the two weeks preceding operation is quite as important as the two weeks following operation.

The day immediately preceding operation the operative field is shaved, a simple enema is given that evening and a good night's rest is assured by means of extra sedative if necessary. The morning of operation the patient is given early morning care and left undisturbed until the pre-operative sedatives are due. What we call a Double Thyroid Sedative is given, i.e., Morphia grains ¼, Atrophine grains 1/150, Hyoscine grains 1/200, one and a half hours before operation; followed by Morphia grains 1/8, Hyoscine grains 1/400 to a woman, and Morphia 1/6, Hyoscine grains 1/400 to a man, and Nembutal grains 1½ three-quarters of an hour before operation. The result of this heavy sedative is that a patient goes placidly to the operating room, usually deeply asleep or at any rate entirely undisturbed by any sense of apprehension.

The post-operative essentials are Lugol's iodine, sedatives and fluids. For this purpose are prepared a sedative enema composed of Lugol's iodine minims 45, Paraldehyde drachms 4. Liquid Paraffin Ounces 3, and an intravenous of Normal Saline. Two ice caps are also prepared. The ether bed is made ready on a Gatz frame without hot water bottles and with no blanket next to the patient.

As soon as the patient returns to the ward the sedative enema is administered, well mixed and heated to body temperature. It is given fairly high as it is expected to be retained and plenty of lubricant should be used, as both Iodine and Paraldehyde are apt to produce painful burns. The Gatz frame is then raised, the ice caps are applied to the head and neck, and the intravenous is started. This intravenous saline is contained in large flasks of 4000 c.c. designed by Dr. R. I. Harris. It is administered at room temperature, that is, it is not heated in any other way, and is given continuosly at the rate of 125 to 150 c.c. an

hour over as long a period as the patient's condition requires; 3,000 c.c. is the usual amount given. Where possible we use a vein low down in the forearm so that the patient's movements are not so restricted as when the hollow of the elbow is used. If the patient is very restless the arm may be controlled comfortably by bandaging it to a pillow. If the patient is not restless a good method is to carry the intravenous tubing down the arm, loop it over the thumb, carry it back up the arm and safety pin a towel snugly over the whole forearm. The patient's pulse is watched carefully from the moment of return to the Ward and a detailed clinical record is kept. Any change in the outline of the neck is noted carefully with the possibility of hemorrhage present; also difficult breathing, particularly difficult inspiration, not expiration as in asthma. We have a routine sedative of Morphine Grains ¼ Atropine Grains 1/150 when required for the forty-eight hours after operation, and this is given sufficiently often to keep the patient resting. If a patient is not restless, but unable to sleep Luminal is administered. During the afternoon and night following operation four doses of Lugol's Iodine minims 10 are administered either by mouth or by rectum. Such cases often suffer discomfort from mucus and difficult swallowing; Liquid Paraffin spray will help this. If mucus is troublesome and not relieved by Liquid Paraffin spray, steam inhalations with Foley Inhaler, using Tr. Benzoin Co. drachms 1 to a quart of water, will often help, and failing this, a steam tent should be used.

The patient spends most of the twenty-four hours after operation in sleep, but has periods of consciousness which are apt to be filled with restlessness and confusion. Her movements are awkward and panicky; if there is a cup within reach she will certainly knock it over; ; when she is given a drink she grasps the tumbler quite unnecessarily, tips the water over herself if possible, chokes and splutters, looks terrified, thinks she wants a bed pan in an awful hurry and suddenly falls asleep again. It is the nurse's care to tide the patient over these stormy moments. Impatience or apprehension of any sort must never be shown no matter what happens. A frightened patient not met by quiet, cheerful reassurance from her nurse suffers intolerably and unnecessarily. Like all post-operative patients, they should be turned often from side to side. These patients hold their necks so stiffly that they often develop a pain across their shoulders. They should be encouraged to allow their muscles to relax and also to speak out clearly instead of whispering. A small pillow lengthwise between the shoulders and up the back of the neck helps the pain by making the patient relax.

The day after operation we remove the large dressing and replace it with a strip of gauze sufficient to cover the wound. On the second day after operation every second clip is removed and on the third day the remainder are removed. If the area around the wound appears inflamed we use a Keith's dressing. If serum collects it is aspirated with a sterile needle and syringe. After three days the average patient is over her troubles. She asks for a mirror and begins to take an intetest in her appearance and also in her meals. This covers our nursing care of the normal uncomplicated convalescence of a patient following a thyroidectomy.

The nurse who is constantly with the patient should be the first to recognize any sign of trouble so that if possible it may be forestalled. A rising pulse, a rising temperature, and a restless patient are always danger signals. They generally mean a patient is heading for "storm." The treatment for storm is a more concentrated form of the routine treatment; more sedative, more fluids and more iodine. A sedative enema composed of Chloral Hydrate grains 20, Potassium Bromide grains 60, Sodium Amytal grains 3, and water ounces 4 is ad-

ministered. Ampoules of Sodium Iodide are available and according to the Surgeons' orders 1 c.c. or 2 c.c. are given every 1-2 hours for 5-10 doses into the tube of the intravenous which has been started. In case of extreme restlessness Sodium Amytal or Paraldehyde may be given intravenously in the same manner. To handle the rising temperature an alcohol sheet is effective and even more so an ice water enema. This is given with two rectal tubes, one high up to expel the ice water, and one low down to drain it off. It is one of the most effective methods of reducing hyperpyrexia that we have used.

Another danger signal to be recognized first by the nurse is respiratory difficulty. This may develop into laryngeal obstruction demonstrated by stridor of inspired air, bluing of the finger tips and a general cyanosed appearance of the patient. On the first sign of respiratory trouble the wound should be examined and if the neck appears full the doctor notified immediately. A tracheotomy set should be always available and if the respiratory difficulty becomes acute should be set up in preparation by the patient's bed. If a tracheotomy tube is installed the opening should be covered with gauze soaked in saline to insure the inhaled air being moist. If the tube plugs, scrub the hands, remove the inner tube, clear it, boil it and replace it.

Tingling in the hands or a tight feeling in the fingers are usually a sign of interference with the parathyroid and should be reported at once. This is generally accommodated by the administration of calcium either in the form of extra milk or calcium chloride in solution which may be taken by mouth or given intravenously.

Pre-operative treatment and operation are only the beginning of the patient's cure. We try to arrange that our patients have at least three months free from responsibility for convalescence; one month to be spent in bed, possibly with bathroom privileges, one month up about the house spending eighteen hours out of the twenty-four in rest; the third month taking short walks and drives but having as yet no responsibility in the home. If there has been severe intoxication then a prolonged bed rest is necessary. In order to avoid painful feet on assuming activity, the patient is required to wear a stout Oxford with low heels; bedroom slippers should never be worn. The patients are sent out with definite written instructions regarding their convalescence. Instructions of course, have to be stretched sometimes to meet the patient's home condition. They are kept on Lugol's Iodine minims 10 a day for six weeks after discharge from hospital and are asked to report back to the Surgeon or to the Thyroid Clinic in the Out Patient Department in three months' time so that their progress may be followed.

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CARDIAC HYPERTROPHY AND DILATATION

By HUBERT HAYWARD, B.A., '37.

More recent trends in physiology have broken down the cenception of the "athlete's heart" and forced us to adopt a new concept of myocardial behaviour. It appears that no amount of activity of the skeletal muscles can cause the myocardium in a normal and healthy circulatory system to undergo changes from which it will not have completely recovered within the period necessary to erase the oxygen debt contracted by the skeletal muscles.

Such facts bring before us forcibly the concept of the immense reserve possessed by the heart. A statement expressing reserve energy as twelve times that expended by the heart of a resting subject probably is not overstating the facts. It is important also to realize that most of the cardiac energy expended in exercise, and that by virtue of its reserve, is the result of increased rate of beat, increased output and raised blood pressure. Note how the skeletal muscles buffer the strain from heavy muscular exercise so that only a fraction of the original comes to play upon the heart at any one time.

If such be the case, then what meaning can hypertrophy have? It is a gruesome one; a sign-post pointing to a chronic pathological condition in the circulatory system. Hypertrophy indicates material enlargement of the cells of the part without interference with their normal arrangements and is to be contrasted with hyperplasia which signifies an increase in the number of cells of the part. Both result in an increase of bulk. These are microscopic distinctions. Macroscopically the term "hypertrophy" covers both, but since hyperplasia of cardiac muscle does not occur except perhaps in lesions in the very young, this term represents the true condition present in the myocardium. Hypertrophy is necessarily associated with increased tissue bulk.

When we think clinically of the enlarged heart another term, "dilatation," comes to mind. The ability of a heart to dilate (physiologically) is its most precious attribute. Every time there is an increase in the work demanded of the heart an efficient response requires that it dilate. The handling of a greater venous return is made more efficient by an increase in diastolic volume of the ventricles, and this requires a diastolic lengthening of the muscle fibres—to which the term "physiological dilatation" of the heart may be applied.

Tonus in smooth muscle, in modern physiology, refers to a property of such tissue whereby it is able to maintain constant internal pressure in a viscus in spite of varying size of content. This property modifies the length of heart muscle fibres at the onset of systole. From Starling's work we understand that the greater the initial length of the fibres, the more forcible will be their contraction. An optimal length exists however beyond which further stretching diminishes the proportional effectiveness of contraction and the heart does not empty itself completely at each beat.

With reference to such events the term "pathological dilatation" has meaning—such occurs when the heart fails properly to discharge its contents when well supplied with venous blood. Such a heart must needs be of low tonus. Starling has further shown that such a heart requires a much more marked increase than normal in its diastolic volume in order to receive sufficient stretching to enable its discharge to keep pace with its inflow.

We are now interested in the cir-

cumstances under which a heart may hypertrophy or dilate (pathologically). Hypertrophy is a process demanding a relatively healthy condition in the fibres in which it occurs. It also does not occur immediately but gradually over a period. Hypertrophy is the response to increased work maintained over a period, and situated within the vascular system, (except in the few cases of adhesive pericarditis). May I emphasize the phrase—"within the vascular system." Such causative factors are best outlined under three headings. (1) Valve lesions whereby obstruction to blood egress or poor expression of output (regurgitation) may occur. (2) Increase in peripheral resistance—an arteriolar phenomenon—may occur. One might argue that this occurs in muscular work, hence might not hypertrophy be related to skeletal muscle activity? However such rise is transient, and not maintained longer than skeletal muscles can maintain their activity. These two classes indicate sources of an absolute increase in the work required of the myocardium. (3) A relative increase in work occurs in myocardial lesions which reduce the amount of muscle tissue without reducing the level of vitality of the remaining fibres to a point at which they are incapable of going through the biological process of hypertrophy. Such lesions result from a local anaemia, local toxicity, or localized bacterial activity in the myocardium.

Pathological dilatation redirects our attention to tonus concepts. Normal tonus maintenance is perhaps the most important index of vitality of cardiac muscle. We can readily understand that general anaemia, generalized toxicity or an acute myocarditis will produce this unwanted effect.

This article is not intended to be a treatise on pathological and physiological processes in cardiac tissue. The writer has but one wish in this regard, that of correlating some pathological and physiological principles relating to cardiac activity.

The author wishes to acknowledge with gratitude the helpful criticism of Dr. Oskar Klotz.

THE TRUE ECONOMY OF DEXTRI-MALTOSE

It is interesting to note that a fair average of the length of time an infant receives Dextri-Maltose is five months: that these five months are the most critical of the baby's life: that the difference in cost to the mother between Dextri-Maltose and the very cheapest carbohydrate, at most is only $6 for this entire period—a few cents a day: that, in the end, it costs the mother less to employ regular medical attendance for her baby than to attempt to do her own feeding, which in numerous cases leads to a seriously sick baby eventually requiring the most costly medical attendance.

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THE DEVELOPMENT OF IMMUNITY TO ANTERIOR PITUITARY HORMONES*

By A. F. GOGGIO, '38.

Work done in the course of the last five years or so by Aron, Loeb and Basset, Siebert and Smith, Loeb and Friedman and others served to show that if experimental animals were treated with various preparations and extracts of anterior pituitary substance at frequent intervals over a considerable period of time, the stimulating effect of these extracts, very evident at first, gradually subsided and the animals no longer responded to such treatment, i.e., they became so to speak, "adapted" or "immune" to the hormonal properties of the extracts. This effect has been shown perhaps most convincingly to be true of the "metabolism-stimulating" property of such extracts which is believed to be due to the thyrotropic hormone of the anterior pituitary gland. We may quote by way of example the concluding words of a paper by Loeb and Friedman on this subject, who state that "After a long continued series of injections of acid extract of cattle anterior pituitary a gradual adaptation, partial or even complete, to the effects of the injection may take place in young guinea pigs and that there seems to be considerable difference in the ability of individual animals to acquire this increased tolerance."

In June of 1933 J. B. Collip came forward with a method for the fractionation of the active principle of the anterior pituitary. He and his collaborators, Drs. D. L. Thomson, Evelyn Anderson and Hans Selye were soon able to obtain a potent thyrotropic fraction, a growth promoting fraction and also an adrenotropic fraction and thus were able to utilize a few of the generally recognized hormones of the anterior pituitary gland in a partially purified state.

Collip and his collaborators began an intensive investigation of the phenomenon of adaptation to anterior pituitary hormones by studying the effect of his purified thyrotropic hormone preparation on normal and hypophysectomized animals. The usual procedure was that of bi-daily intra-peritoneal injections. They observed that their purified thyrotropic extract caused a marked hyperplasia of the thyroid gland with an accompanying increase of metabolic rate in the normal white rat, the hypophysectomized rat and the guinea pig. But this stimulating effect did not persist; the metabolic rate in spite of continued injections returned to normal within 2 or 3 weeks, the thyroid gland also showing signs of returning to normal. Moreover, and what was more remarkable, they were able to demonstrate the fact that if the injections were continued for a long enough time the metabolic rates of normal animals after the invariable preliminary rise might fall below the normal level, in some cases as low as the metabolic rate of untreated hypophysectomized animals, i.e., about 26% below normal in the case of rats.

In the case of these normal rats whose metabolic rates had fallen below normal as a result of long continued administration of the thyrotropic extract, Collip and co-workers made the following observations:—

(1) Increasing the dose of thyrotropic hormone preparation as much as 8 times gave no rise in metabolic rate.

*Presented to the Physiological Journal Club, Jan. 8, 1935.

(2) A thyroid transplant from a normal animal gave no increase in metabolic rate. On the other hand when these thyrotropic resistant rats were fed small amounts of dessicated thyroid tissue there was observed a sharp rise in metabolic rate. Thus it appeared that the low metabolic rate of these animals was due to some inhibiting effect on the activity of the thyroid gland and not to any developed immunity to the potent product of the gland,—thyroxin.

(3) The most significant observation of all was the following:—Serum taken from these resistant animals which had received long treatment with thyrotropic hormone, on being given twice daily to hypophysectomized rats together with the usual daily injection of the active thyrotropic principle, entirely inhibited the thyrotropic effect of the latter. So much so that, even if the dose of thyrotropic preparation given was as much as 200 times the effective dose, the usual rise in metabolic rate was noticeably inhibited. By way of control they showed that normal rat serum had no inhibiting effect on the action of the thyrotropic hormone.

In April, 1934, Collip and Anderson published a paper entitled "The Preparation and Properties of an Antithyrotropic Substance" whose content was essentially the following:—A 15 year old mare whose serum before treatment had no effect on the action of thyrotropic hormone was given daily intravenous injections of Collip's thyrotropic hormonic preparation. After four weeks of such treatment the horse's serum began to show definite antithyrotropic properties; when administered to hypophysectomized rats, 2cc. of serum completely inhibited a dose of thyrotropic hormone of 10 times the minimum effective dose. The inhibitory potency of the serum was found to be increased by giving the horse progressively larger doses of thyrotropic hormone over a period of 6 weeks or so. Thus a horse serum was produced which had to a very marked degree the antithyrotropic properties which we have described in the case of the serum of thyrotropic resistant rats.

The question then arose as to whether or not the effectiveness of this antithyrotropic serum was due to some known type of immunological reaction with the thyrotropic hormone. It was found that no positive complement fixation or precipitin tests could be obtained, thus indicating something other than the usual immunological reaction in which the protein of the serum (globulin fraction) plays an important part. Collip and his collaborators therefore went on to attempt to prepare serum-protein-free extract of this antithyrotropic blood, and this they soon succeeded in doing.

They then proceeded to test this protein-free antithyrotropic extract against thyrotropic hormone in normal rats. They found their extract to have very active antithyrotropic properties. Not only was the action of the added thyrotropic hormone completely inhibited in the case of normal rats treated with both antithyrotropic and thyrotropic preparations simultaneously, but further, the rats' own thyrotropic hormone was also inhibited, in one case the metabolic rate being lowered to 26% below normal—i.e., the level of the hypophysectomized rat. In hypophysectomized rats the antithyrotropic potency of the serum-protein-free extract was similarly demonstrated.

In November, 1934, Scowen and Spence of London, England, in a report of work they had been doing on anterior pituitary hormones, confirmed the work of Collip and his collaborators. They did this by demonstrating the production of an antithyrotropic substance in the serum of rabbits receiving prolonged treatment with anterior pituitary extract. They suggested that the antithyrotropic principle might be formed by the suprarenal glands as a result of the action of the adrenotropic factor which in their case was of course ad-

ministered along with the thyrotropic factor in the anterior pituitary extract they used. However, in view of the claim of Collip and Co. that their adrenotropic extract is practically free of the adrenotropic principle, this suggestion perhaps does not carry much force.

But to consider further the effects of prolonged administration of thyrotropic hormone on the thyroid gland and on the metabolic rats of test animals. Collip and his collaborators went on to investigate the changes, if any, produced by this treatment on the pituitary glands of test animals. To do this they took the pituitary glands of chronically treated and therefore thyrotropic resistant rats and by implanting them into young hypophysectomized rats were able to show that the pituitary glands of these resistant animals contained no thyrotropic hormone. That the growth hormone was still present in these glands was shown by the fact that the young implanted rats increased in body weight during the experiment.

Summarizing briefly therefore the main results of this work, it appears that long continued injection of thyrotropic hormone results in the appearance of an antithyrotropic substance in the serum and in certain organs of the treated animal and also results in the suppression of thyrotropic hormone formation in the anterior pituitary lobe of the animal.

Where in the body the antithyrotropic substance is formed is not yet known. The site of its formation does not seem to be in the hypophysis, or at least not exclusively there, for hypophysectomized rats on prolonged treatment with thyrotropic hormone become resistant to it in the same way that normal rats do.

Collip and his associates naturally began searching for similar "anti-hormone" effects, as they called them, in the case of other anterior pituitary hormones. In this research they have met with some success, but due to the extreme recency of the work, the reported results are as yet very meagre.

It is claimed that an anti-growth effect has been quite successfully demonstrated. Young hypophysectomized rats on prolonged administration of Collip's growth hormone preparation ceased after a time to put on weight and actually showed a decline in the growth curve in some cases. The serum of these growth-hormone-resistant animals when administered together with growth hormone preparation to other hypophysectomized rats was observed definitely to inhibit the action of the growth hormone. Furthermore an active anti-growth serum was prepared by chronically treating a horse with the growth hormone preparation.

Collip and his collaborators have also reported work which is indicative of anti-diabetogenic and anti-ketogenic effects of a similar nature to the anti-thyrotropic and anti-growth effects here discussed, but their evidence is as yet far from conclusive. Little has been accomplished too with regard to the gonadotropic hormones of the anterior pituitary gland although Collip has been able to show that rats become non-responsive to the "maturity factor" present in fresh anterior pituitary gland on repeated daily administration of this substance. Aron, in the case of guinea pigs, has also reported a loss of sensitivity to pituitary gondaotropic extracts after prolonged treatment. No evidence has yet been obtained concerning any possible anti-adrenotropic or antithyrotropic effects.

Such, then, are the most important facts concerning the development of immunity to anterior pituitary hormones as investigated thus far. Perhaps it would be of interest to consider the interpretation put on these phenomena by J. B. Collip who, as a result of all his work in this field together with a consideration of the work of others, has been led to formulate tentatively what we may call his "anti-hormone hypothesis." The essence of this hypothesis is the assumption of the elaboration and normal presence in animals of both hormones and anti-hor-

mones, the anti-hormone acting as the antagonist of the corresponding hormone. Collip suggests that the anti-hormone content of an animal is directly related to the level of the corresponding hormone, and that it is the ratio of the two which is important in determining the effect produced. Since the presence of the anti-hormone would tend to allow an increase in the amounts of hormone (and anti-hormone) present in the animal, Collip suggests that this and the ratio effect just mentioned might account for the great differences in responsiveness to hormone preparations observed in different species and in different individuals of the same species. He suggests that we may regard some animals as being better "buffered" with respect to a particular hormone effect by having in their bodies larger amounts of both hormone and anti-hormone than other animals. Obviously, then, the injection of a given amount of hormone will have less effect on the former well-buffered animals than on the others. Quite in keeping with these theoretical considerations is Collip's general law of "inverse response" which he derived from direct experimental observation. He states, "The sensitivity of an animal to injected hormone varies inversely with the hormone content or production of the animal's own gland."

Thus, if we consider by way of example the frequently observed case of the rat, we find the following situation. The production on hyperthyroidism in a normal rat by the injection of thyrotropic hormone is difficult (as compared with guinea pig say), i.e., the sensitivity of the rat to thyrotropic hormone is relatively very low. On investigation we find that the anterior pituitary gland of the normal rat is relatively rich in thyrotropic hormone. Our explanation thus appears quite reasonable, namely, that the normal rat's content of both thyrotropic and antithyrotropic substances is high, that he is therefore well "buffered" against the effects of changes in thy rotropic hormone, and that the addition if a small amount of thyrotropic hormone by injection is unable to change appreciably the normal ratio to thyrotropic to anti-thyrotropic substance in the rat's body and thus is unable to exert any appreciable stimulating action of the thyroid gland. The guinea pig, relatively very sensitive to thyrotropic hormone, is found to have a low thyrotropic content in his own gland. The case of the hypophysectomized animal is also interesting. Collip claims to have shown a progressive increase in sensitivity to thyrotropic hormone after hypophysectomy which attains a relatively extreme degree. The interpretation of this would be that the cutting off of thyrotropic hormone production results in a rapid fall in the thyrotropic level of the animal; this brings about progressive falling off of the antithyrotropic level with a consequent increased sensitivity to the thyrotropic hormone.

However interesting as this hypothesis may be, it is, of course, by no means established as yet, and Collip himself, as a result of further work, may modify his views considerably within the next few years. We must therefore summarize what has been learned concerning the development of immunity to anterior pituitary hormones more or less cautiously. We may say that an immunity to anterior pituitary hormones does develop when these hormones are injected over a prolonged period of time; we may say that the immunity developed is apparently a specific immunity to the particular hormone injected; and we may say further that it appears to be due to the appearance in the treated animal of some sort of neutralizing substance which J. B. Collop believes to be an "anti-hormone."

THE SYMPTOM --- DIARRHOEA

By TREVOR OWEN, M.B., M.R.C.P.

To the layman, diarrhoea may mean anything from sprue, or an acute gastro-entero-colitis with numerous stools, to a frequent or a persistent desire to empty the rectum, due to the irritation of a fissure-in-ano, for example—whether any evacuation takes place or not. It is, therefore, essential to ascertain what he really means by the term.

In considering this symptom, one must keep in mind the complex "mechanism" which governs the motility of the gastrointestinal system, so far as we know it, and eliminate one by one each particular factor, abnormalities of which alter the muscular action in such a way as to produce frequent evacuation. Briefly, and in a general way, normal activity depends on: (1) normal nervous control, (2) a balanced flow of normal blood and lymph, (3) contents having a normal physical and chemical nature and suitable for that particular organism, and (4) a healthy wall (a) mucous membrane, (b) muscular coat and (c) peritoneum. Any alteration in one or more of these factors may produce abnormal motility of the whole or a part of the tract.

Diarrhoea from Altered Nervous Control.

No organic spinal nervous disease, of itself, causes diarrhoea. The intestinal tract is supplied by the splanchnic nervous system, a complex of spinal and sympathetic fibres. Of diseases of this "system" almost nothing is known, yet quite conceivably lesions there would disturb the motility of the tract. So far, diarrhoea of this origin has not been diagnosed.

At either end of the intestinal tract this involuntary nervous control is replaced or augmented by voluntary nervous control, so that diseases of the motor and sensory tracts of the latter will alter the control of the rectal sphincter. In motor and sensory lesions, there will be incontinence of faeces which might be complained of by the patient as "diarrhoea." In Tabes Dorsalis the so-called rectal "crises" occur. There is an intense desire to empty rectum but the patient is unable to do so.

A few cases of chronic intermittent diarrhoea are probably due to abnormal function of the vaso-motor system ascribable in some manner to toxaemia from a low grade infection. An increased excretion of water into the intestine with hyperperistalsis leads to evacuation of watery stools. A similar "mechanism" may also be responsible for periodic diarrhoea occurring in migrainous patients.

Among the vaso-motor phenomena can be placed the diarrhoea experienced during excitement, fright, fear and from exposure to severe cold. These conditions have, however, not been proved to be of this nature, but may rather be due to a transitory physiological hyperadrenalism or hyperthyreoidism, or both.

Certain drugs acting on the nerve endings produce hyperactivity of the gut (e.g. physostigmine).

In the so-called functional diseases of the nervous system diarrhoea may occur. The actual mechanism of its production, whether by abnormality in the nervous impulses or secondary to alteration in the secretion of the digestive juices, is not understood. In hysteria, diarrhoea is not uncommon. Mucous colitis is classed by some as a functional nervous derangement, in which diarrhoea may alternate with constipation with the passage of large or small quantities of mucus not containing pus or blood.

Diarrhoea Due to Vascular Disease.

A. **Arterial**: In the arteriosclerotic, a chronic diarrhoea may be present, but is far more rare a symptom than is constipation.

Embolism: The mesenteric arteries constitute one of the rarer sites for embolism. The interference with the blood supply leads to oedema of the part with haemorrhage and an early symptom is transitory diarrhoea with free blood in the discharges.

Thrombosis of an artery is accompanied by great shock which depends on the size of the branch affected as also does the occurrence of diarrhoea with bloody stools; the larger the artery the less likely it is to be a symptom because of the death of a section of bowel, leading to symptoms of intestinal obstruction.

B. **Venous**: Thrombosis of the mesenteric veins occurs secondary to portal thrombosis or to compression by enlarged mesenteric glands, pancreatic or gastric tumours, or by local peritonitis due to gall bladded disease or peptic ulcers. Primary thrombosis occurs in the branches of veins from any ulcerated or inflamed area in the bowel, especially in the terminal ileum, appendix or caecum. In pylephlebitis, the diarrhoea may be paroxysmal. Diarrhoea is a symptom in about 50 per cent. of the cases of venous thrombosis. The onset is characteristically sudden with severe generalized colicky pain and if diarrhoea occurs, the stools are bloody. Constipation and symptoms of intestinal obstruction follow.

Passive congestion from myocardial failure, if severe, and especially if of long duration, causes diarrhoea probably from oedema of the intestinal walls.

Diarrhoea from Obstruction of Lymph Flow.

In obliterative lesions of the retroperitoneal lymph glands (e.g. Tabes mesenterica) the absorption of fat may be completely prevented and produce a chronic diarrhoea. The same phenomena may occur in obstruction of the thoracic duct.

Diarrhoea Due to Changes in the Quality of the Blood.

Alteration of the haemoglobin content or in the number of red and white cells does not, in itself, account for the symptom of diarrhoea occurring in such cases. In the leukaemias, infiltration of the wall and particularly of the lymphoid tissue by the abnormal leucocytes may provoke reaction in the wall as in any other new growth. In methaemoglobinaemia and sulphaemoglobinaemia, a chronic diarrhoea is often associated. It is to abnormalities of the plasma that this heading refers. All forms of toxaemia, organic and inorganic, and exogenous or endogenous in origin, are included in this group. The intestinal tract is a pathway for the excretion of all toxins and therefore those of all acute infections (e.g. diphtheria, bacillary dysentery, cholera, etc.), inorganic poisons (e.g. mercury, arsenic), organic endogenous toxins (e.g. renal insufficiency, liver insufficiency, acute gout); all may produce diarrhoea by injury to the wall when concentration is great. This is an added factor in those diseases (e.g. bacillary dysentery) where the primary lesion also is in the intestinal tract. The diarrhoea of hyperthyroidism is probably a direct action on the muscle from an increased "thyroxin" content of the blood.

Diarrhoea Due to Abnormal Quality of the Contents.

Mineral oil in excess and laxative or purgative substances can be mentioned under this heading, though some not strictly so. The absence of bile in obstructive jaundice and of the pancreatic secretion in obstruction to the pancreatic duct gives rise to incomplete digestion of fats, and the irritation due to fatty acids may cause diarrhoea. Achylia gastrica is frequently the apparent factor in a form of looseness of the bowels taking place after any food.

In some individuals diarrhoea is an allergic phenomenon after partaking of certain foods. Egg, milk and shellfish are the most common. While nearly all persons of this nature are early aware of their peculiarity, the presence of the harmful ingredient may not be apparent in the dish eaten.

The toxins of deteriorated or infected foods, spoken of under the name "ptomaines," are also not an infrequent cause of local epidemic diarrhoea.

Diarrhoea from Diseases of the Wall of the Intestinal Tract.

Infections specifically attacking the intestinal wall cause hyperperistalsis by inflammatory reaction with or without ulceration, and have diarrhoea as one of the chief symptoms. It is not the purpose here to describe the clinical characteristics of these diseases (ameobic and bacillary dysentery, tuberculosis and syphilis of the intestine, typhoid and paratyphoid fever, ulcerative colitis, cholera, fungus infections, leprosy).

Inflammatory or degenerative changes not due primarily to infection are seen in pernicious anaemia, pellagra, sprue, amyloid disease, scurvy.

New growths, polyposis, diverticula (the appendix is a diverticulum) and peritoneal disease, cause diarrhoea either by mechanical irritation of by ulceration and secondary infection, or by haemorrhage. It is often of an intermittent type.

Symptoms Indicating the Part of the Tract Infected.

The higher up in the gut the abnormalty is, the more will the diarrhoea be related to, or exaggerated by, the taking of food (e.g. achylia gastrica), or be accompanied by vomiting or nausea (e.g. acute enteritis).

Hyperperistalsis of the small bowel, if severe, produces a generalized colic with sharp "narrow" pains.

When the colon is affected there is little or no change in relation to taking food. Colic, if present, is felt as a "broad" pain more likely to be localized than that arising from the small intestine. There may be regional tenderness.

As the site of the lesion approaches the rectum, tenesmus becomes a more prominent symptom. Naturally, if the whole tract, from stomach to rectum, is involved, all these symptoms may be present together.

The Information Gained from Examination of the Stools.

(a) Regarding the Position of the Lesion. Watery stools arise in the small intestine. Bright red blood is usually from the sigmoid colon, or rectum and the nearer the lesion is to the ileocaecal valve the darker red will the blood be. Blood from the small intestine is changed still more and that coming from the duodenum is tarry.

Large amounts of mucus probably indicate the small bowel, and small amounts the colon, as being the affected part.

Very acid, sour smelling, light coloured and "crumbly" stools are associated with small intestinal lesions due to abnormalities in the digestive processes.

Undigested food particles, especially meat, are present in small intestinal diarrhoea (lienteric diarrhoea).

Very small discharges indicate a rectal lesion.

A long period of constipation followed by the evacuation of enormous stools is pathognomonic of Hirschsprung's disease (megacolon).

(b) Regarding the Kind of Lesion.

Pus means ulceration.

Blood cells means congestion, but gloss blood means ulceration or simple rupture of a vessel, purpura, thrombosis, embolism, or new growth.

The specific organism can often be found at the proper time in tuberculosis ,bacillary and amoebic dysentery, typhoid and paratyphoid fever in parastic infestations and the fungus infections.

An attempt has been made to consider the question of diarrhoea as an example of the method which should be and can be employed in working out the origin of each symptom of any symptom-complex. The omission of any, or a change in the order, of the various steps will often result in a general sense of bewilderment. The investigation of "diarrhoea" will emphasize the fact that short cuts to diagnosis are short cuts to failure.

THE EXAMINATION OF THE NERVOUS SYSTEM

By H. H. HYLAND, M.D.

It is essential in the examination of the nervous system that a systematic procedure is used which will be employed fully on every neurological patient. Unless the system of examination is firmly fixed in the mind of the examiner much time will be wasted and important points missed.

The actual order of examination is not of great importance and may be left to the discretion of the individual, but whatever order is adopted should be followed faithfully in every case. If this is done it is unlikely that omissions will occur.

The following is a brief outline of a system of examination containing the minimum necessary for a satisfactory investigation of the nervous system.

General Impression of the Patient.

The attitude of the limbs and the head are observed, the presence of involuntary movements (choreiform, athetotic, tremors, etc.); the expression of the face (immobility, grimacing, tabetic mask, etc.).

Higher Mental Functions.

Is coma or delirium present? Is the patient unduly drowsy? Is he attentive or does his mind wander? Is he negatavistic or unduly suggestible? Is he co-operative? Is orientation good in space and time? Is the memory good for recent and remote events? Is he euphoric or unduly depressed? Is there flight of ideas or retardation of thought? Question carefully for delusions or hallucinations.

If there is reason to suspect mental impairment, ask question in mental arithmetic and submit questions of current interest. The answers will indicate defects in perception and judgment which may be present.

From the above examination an impression may be gained of the patient's mental status. More detailed examination will be necessary of there is reason to suspect a psychosis or gross mental impairment.

If spontaneous speech is affected the possibility of dysarthria, or aphasia, must be considered.

Dysarthria (articulatory disturbance) is made more apparent when the patient repeats test phrases, e.g., "Truly rural," "British constitution," etc. If the muscles and peripheral nerves of the articulatory mechanism are intact, dysarthria is due either to disease of the pyramidal system (as in G.I.P.) or of the cerebellum (as in disseminate sclerosis) slurring, omission of syllables, reduplication of syllables will be noted.

Aphasia (non-articulatory disturbance) must be clearly distinguished from dysarthria. The articulatory mechanism is intact but the exteriorization and comprehension of written and spoken speech are affected.

A useful routine method for examining aphasic patients follows:

Is the patient right or left handed? What education has he had previously? Does he understand the nature and use of common objects? Can he understand gestures? e.g., make a movement as if

to strike him and note his reaction. Is his hearing intact,—if not how much disturbed? Can he recognize ordinary sounds and noises? Can he comprehend spoken language? e.g., does he obey commands at once—what mistakes? What is the nature of spontaneous speech? Does he omit words, use wrong words, mutilate words, construct his sentences faultily or talk jargon? Is perseveration of speech present? e.g., does he continue repeating one phrase or sentence in answer to several different questions? Can he repeat words uttered in his hearing?

Test automatic speech by having patient say the Lord's Prayer or the alphabet, etc.

Be sure the eyesight permits reading and then test ability to comprehend written speech. Can he obey written commands? Does he recognize words, letters or numerals? Can he write spontaneously — what mistakes? Can he copy written words or write from dictation? Can he perform simple written arithmetic problems? Can he read aloud? What mistakes? Does he understand what he needs? Can he name at sight common objects? When several objects are presented to him can he match each one with its name spoken or written? Can he still play cards or play a musical instrument?

Test the patient for apraxia by handing him familiar objects, e.g., tooth brush, match and cigarette, telephone, etc.

When the results of this examination are all before the examiner he must carefully decide the character and degree of the aphasia present. In all aphasias the speech function is depressed as a whole and there is a general lowering of the higher mental faculties, but it is important to know if the aphasia is mostly motor, or mostly sensory, if word blindness is present or word deafness, etc. The examination should never be done at one sitting, but on several occasions because aphasics tire quickly and the aphasia often varies from day to day.

Cranial Nerves.

Olfactory Nerve.

Only those substances are appreciated which are readily oxidizable, therefore only the essential oils should be used in testing, e.g., oil of cloves, oil of wintergreen, etc.

Test each nostril separately and remember that there is a large normal variation between individuals in the keenness of sense of smell.

Unilateral anosmia is more important than bilateral—the latter may be produced by several local and general diseases.

If anosmia is found it is usually of value to determine that the 5th nerve is intact and that the anosmia is not due to an atrophic process in the mucous membrane. This is done by observing the reaction of the patient when told to inhale spirit of ammonia on the affected side. If the reaction is normal it indicates that the anosmia is due to a lesion of the olfactory nerve.

Optic Nerve.

Acuity of vision. This must be tested in all neurological cases using Snellen's charts. It is done without glasses (uncorrected) and with glasses (corrected) and the results charted.

Visual fields. — The rough test is usually employed. The right eye of the patient is fixed by the left eye of the examiner and the other eye closed. A small round white object should be used and brought in from the periphery by the examiner from all angles. If the patient's field is more restricted than the examiner's the test must be repeated accurately by using a perimeter. The other eye is examined in the same fashion.

Certain patients are unable to cooperate in this test, and it may be of the greatest importance to determine if a field defect is present. In these cases if the examiner stands behind the patient while someone endeavours to attract attention in front, threatening movements in view of the temporal fields on each side may indicate the

side of the defect by the absence of the blinking reflex.

The most important field defects to be recognized are right or left homonymous hemianopias, bitemporal hemianopias and quadrantic field defects.

Scotomata—can be readily tested by employing a small white object attached to a long stalk. The old-fashioned hat pin with a head as large as a pea is satisfactory. The eye of the patient is fixed as in testing the visual field and the object slowly moved from the point of central vision towards the periphery in all directions. The examiner measures his own blind spot against the patient's and any increase in size of the patient's blind spot is noted. Time and care are necessary in getting results and the co-operation of the patient is necessary. Colored objects should be used in addition to white—red and green are the most important. In retrobubar neuritis a central colour scotoma is one of the earliest signs.

Ophthalmoscopic Examination.

In the retina the most important observations from a neurological standpoint are changes in the macula (e.g. cherry red spot in amaurotic family idiocy), vascular abnormalities (e.g. haemorrhage, arteriosclerosis), and syphilitic choroiditis.

In the examination of the fundus we look for papilloedema and often atrophy. Papilloedema, if well marked, is easy to recognize, but the very early changes are often overlooked. It is well to remember that the nasal side of the disc is the first to show change, it becomes less distinct and the whole disc becomes increasingly red with disappearance of the physiological cup.

Vision may be perfect with high degrees of papilloedema present, particularly if the latter is of acute onset. When papilloedema has been present for some time, however, optic atrophy commences and visual failure occurs proportionately.

Optic Atrophy may be divided into 3 types: (1) primary, (2) secondary, (3) consecutive.

(1) Primary is due to original devitilization and death of the nerve cells in the retina—the disc is pure white and flat, the vessels are small, the lamina cribrosa is plain, the edges of the disc are sharply cut.

(2) Secondary—results from lesions of the optic chiasma or the optic nerve —the disc may exactly resemble that found in primary optic atrophy or some slight irregularity of the margins may be present.

(3) Consecutive—follows severe papilloedema. The disc is white and flat but the edges are fluffy and irregular.
Oculomotor Nerves (3, 4 & 6).

Ptosis, if present, will be noted first. A true ptosis must be distinguished from a "pseudoptosis" due to lesion of the cervical sympathetic causing of paralysis of the involuntary component of levator muscle. The latter produces a narrowing of the palpebral aperture on the affected side, but the upper lid does not cover the pupil when the patient looks directly in front.

Enopthalmos and exophthalmos are looked for.

Eye movements are next observed. The eyes are examined at rest and any strabismus noted. Then the patient is requested to follow an object with his eyes, keeping his head motionless. The object is moved to the extreme right and left and up and down. If a strabismus is present but no double vision occurs (visual acuity being good in both eyes) it can be assumed that the ocular paralysis is of long standing and that the patient has learned to suppress the image seen by one of the eyes. If diplopia occurs in the presence of a well marked squint, slight or not recognizable, it can be assumed that the affected eye is the one on the same side as the diplopia. A red glass is placed over one eye and a light moved around through the field of vision. When diplopia occurs the position of the red and white images is noted. If the red image is on the opposite side

to the covered eye (the white image will be similarly situated in relation to the uncovered eye) a crossed diplopsia is present. If the red image is on the same side as the covered eye the diplopia is uncrossed. Crossed diplopia occurs with divergent strabismus, uncrossed with convergent strabismus. As the object is moved about the relative positions of the images are noted. With a knowledge of the functions of the ocular muscles it can be concluded which are at fault.

We have been considering the effect of peripheral impairment of ocular movement, but mention must be made of defects in conjugate deviation of the eyes which occur with supranuclear lesions of the oculomotor nerves.

Paralysis of conjugate deviation is not in terms of muscles but of movements. Thus you may see paralysis of upward movements of the eyes, paralysis of convergence, paralysis of lateral movements to the right, or left, and paralysis of downward movement. There is no strabismus or diplopia with defects in conjugate deviation, and it is important to distinguish this type of ocular paralysis from that resulting from disease of the oculomotor nerves of the lower motor neurone type.

The pupils are examined in regard to size, inequality, shape, contour, reactions to light, direct and consensual, and reaction to accommodation.

In testing for light it is important to always use the same strength of illumination thus providing a standard in judging sluggishness of pupils. It is necessary always to fix accommodation, preferably on some distant object, before throwing light into the pupil.

Nystagmus will be seen if present when the eyes are at rest. The patient follows an object with his eyes to the right and to the left. If nystagmus occurs on lateral deviation to the right or left or both, note its character and rate. It is usually horizontal but may be vertical or rotatory. It may be coarse or fine, slow or rapid.

True nystagmus is well sustained and lasts indefinitely. This must be distinguished from nystagmoid jerks, which are usually of little significance and can be obtained in many people if the lateral deviation is very extreme or the patient tired.

Trigeminal Nerve.

Motor division The patient is asked to open his mouth against resistance. Any deviation of the jaw is noted; if the relative position of the front teeth is observed before the patient opens his mouth the examiner will not be deceived in the event of a facial palsy being present also.

In paralysis of the motor division the jaw deviates to the side of the lesion. The muscles on the floor of the mouth fail to harden on the side of the lesion. The patient is asked to bite and the anterior edges of the masseter muscles are felt at the time of the movement. The temporal muscles are also felt and any weakness in temporals or masseters will be readily detected as the jaws meet.

If the patient moves his lower jaw sideways with the mouth open there will be diminished excursion to the opposite side if a lesion is present.

Sensory Division. The corneal reflexes are tested with a horsehair after ascertaining that both orbiculares oculi are functioning well. A difference on the two sides may be of great importance.

Sensation is tested over the area supplied by the three sensory divisions of the nerve, using cotton wool, heat and cold, and pin prick.

N.B.—In Tic Douloureux no sensory loss is present but the skin over the area of pain may be hyperiesthetic and tender points will be found where the main branches come close to the surface.

Facial Nerve.

Any asymmetry of the face is noted while at rest. The patient is asked to show his teeth and any failure of the two sides of the mouth to move out equally is observed. Just as important is an asymmetry in the furrows ex-

tending from the lateral borders of the nose.

In slight facial weakness the mouth may be drawn out well on the affected side but the corner of the mouth will not be elevated properly and will be on a lower horizontal plane than its fellow on the opposite side. The furrow will be less prominent and shallower on the weak side.

The patient is asked to "screw his eyes up tight" and not let the examiner open them. A weakness in the orbicularis oculi is readily detected by this means.

Additional information may be obtained by asking the patient to frown, wrinkle his forehead, whistle and blow out his cheeks.

The effect of emotional movement must then be observed, e.g., smiling, etc.

In lower motor neurone paralysis all movements are affected, voluntary and involuntary, the upper and lower part of the fact are equally paralyzed on the side of the lesion.

In upper motor neurone paralysis the lower part of the face is much more affected than the upper part and involuntary movements, e.g., smiling, show no weakness.

In thalamic lesions involuntary movements only are affected, voluntary movements being normally performed.

In lower motor neurone lesions the sense of taste on the anterior two-thirds of the tongue should be tested. It is lost in lesions peripheral to the geniculate ganglion, the chorda tympani being involved in the lesion.

To test taste the tongue is protruded and dried. Minute quantities of salt, sugar, vinegar and quinine are placed on the affected side of the tongue with an applicator. After each substance is placed on the tongue it is wiped off before the patient puts his tongue in his mouth, otherwise the substance will pass to the normal side and the patient's statement will be valueless.

Auditory Nerve.

Cochlear Division. One may test hearing with a watch, doing each ear separately and keeping closed the external auditory meatus of the ear not being tested. One must know what to normally expect with the watch that is used and the test must be performed in a quiet place.

The most important finding is a difference on the two sides and if this is found otoscopy should always be done.

If deafness is present it should be determined whether it is due to a lesion of the cochlear nerve or to middle ear disease.

Rinne's Test: A vibrating tuning fork is applied to the mastoid process on the affected side. When the patient ceases to hear it in that position the vibrating end is held close to the ear. In nerve deafness the hearing by air conduction and bone conduction is completely lost or else the patient hears by air conduction after he has ceased to hear by bone conduction. In middle ear deafness hearing by bone conduction lasts long after it has ceased to be audible by ear.

Weber's test: A tuning fork is applied to the centre of the forehead. In middle ear disease the sound is heard best on the deaf side. In nerve deafness it is heard best on the sound side.

Vestibular Division. Lesions of this division produce symptoms almost identical with those of cerebellar disease. Nerve deafness is always present, however, and the cerebellar symptoms are only seen in vestibular lesions when the condition is acute.

Vestibular functions are examined by the galvanic test, Barany's caloric test and the rotating test. Normally, following these tests, nystagmus, deviation of the eyes and "past-pointing" occur; but if the vestibular mechanism is at fault the normal response is altered.

Glossopharynegeal and Vagus Nerves.

These nerves are considered to-

gether because their functions cannot be clearly separated.

The patient is asked to open his mouth and the palate is observed. If a lesion be present the palate may be seen to hang lower on the affected side. When the patient says "Ah" the palate moves up towards the non-paralyzed side. In bilateral lesions it will not move at all.

Sensation of the palate and pharynx should be tested. In unilateral lesions there will be anaesthesia of the palate and pharynx on the affected side, as shown by the absence of reflexes when the parts are tickled.

Difficulty in swallowing is never severe unless the lesion is bilateral, but a moderate difficulty may be present with unilateral lesions more marked for fluids and solids.

The condition of the voice is noted, particularly a nasal tone, hoarseness or stridor. The larynx should always be examined if any evidence of paralysis of the tenth nerve is present whether changes in the voice are present or not.

Spinal Accessory Nerve.

Note whether any winging is present at the angle of the scapula.

The patient is asked to shrug the shoulders against resistance and any difference on the two sides is noted.

If doubt exists several other tests for the trapezii may be used, e.g., approximating the scapulae behind, and carrying the extended arm backwards.

The sternomastoids are tested by having the patient turn his head to the right and left while the examiner offers resistance with one hand and palpates the contracting muscles with the other. The affected muscle does not harden when the head turns to the side opposite the lesion.

Hypoglossal Nerve.

The patient is asked to protrude the tongue. In the presence of a unilateral lesion the tongue will deviate to the paralyzed side, being pushed over by the healthy muscles.

Atrophy may be seen on the affected side; the tongue is smaller and the mucous membrane has a crenated or wrinkled appearance on that side. In advanced atrophy the tongue is sickle-shaped.

In bilateral lesions (bulbar) fibrillation may be seen in the atrophied muscles.

Motor System.

Head and neck—the position of the head and any wasting of the muscles is noted. Passive movements and active movements against resistance are tried and any limitation or weakness observed.

Upper limbs—the following points are observed:

(1) Position of limbs at rest.

(2) Wasting — if present is it confined to muscle groups or is it generalized? The small muscles of the hands and the muscles of the shoulder girdles are the first to show wasting as a rule. If wasting is present look for fibrillation.

(3) Muscle power — the power is tested at all joints; the patient is told to perform the given movement against resistance and any weakness or inequality on the two sides noted. He is then told to hold the joints immobile and resist the examiner's attempt at movement.

Any difference in the grip of the two hands is noted. Normally the right hand will be a little stronger than the left if the patient is right handed; a dynamometer should always be used. If any indications are present the power in the fingers must be tested separately for flexion and extension at all joints and in abduction and adduction.

(4) Passive movement is tested at all joints and any limitation of movement or alterations in tone noted.

(a) Hypertonia—before concluding that tone is increased flexion and extension must be repeated several times (requesting the patient to relax) in order to rule out any voluntary element. If the limb is persistently hyper it must be decided whether it is pyra-

midal (hemiplegia) or extra-pyramidal (parkinsonism) in type.

If the latter it is equally present through the whole range of movement and probably accompanied by the cog-wheel sensation. If the former it is of the clasp-knife variety and much more marked on attempting extension than flexion.

(b) Hypotonia is often difficult to determine. If increased mobility is present in the joints and the limbs can be placed in abnormal positions it is easy, but in early cases this is not so.

Several useful tests are available for detecting hypotonia.

Stand behind the patient supporting his outstretched arms with your hands and give the arms little throws upwards. The hypotonic arms falls more heavily on the hands.

Hold the patient's hand with the arm flexed at the elbow and tell him to pull hard; suddenly let go his hand and if the arm is hypotonic his hand will hit his face. This test is rather brutal but very satisfactory.

Ask the patient to relax his arm and suddenly throw it at his face—if the arm is hypotonic he will not stop it in time and it strikes the face. The normal arm will not do so.

(5) Involuntary Movements. Any choreic or athetoid movements or tremors at rest will have been noted. Certain tremors are present at rest and abolished by movement (e.g., paralysis agitans) other tremors only appear on movement (e.g. disseminate sclerosis).

(6) Co-ordination — severe inco-ordination will be obvious without special testing. The patient has difficulty in picking up objects, in buttoning his clother and in feeding himself with the affected hand. Two tests are used to demonstrate the disturbance.

Finger-nose test. With the eyes open the patient is asked to touch the tip of his nose with the forefinger of the affected hand. It is important that the patient's arm is free from support during this test and that the elbow does not touch the bed. The movement should be performed slowly. Inco-ordination is recognized by the hand deviating in an uncertain fashion from the direct course to the nose and the finger strikes the face or the bridge of the nose. The test is also useful to demonstrate intention tremor. In early cases this will be seen at the end of the movement and the finger after striking the nose slips off or it may flatten the nose several times before coming to rest Well marked cases of intention tremor show themselves earlier in the movement and increase as the objective is approached.

After the test has been performed with the eyes open it is permissible to have it done with the eyes closed, but it must be remembered that this introduces a new element, e.g., sense of passive movement.

Finger-nose-finger test—the examiner holds his finger up and the patient touches it with his forefinger and then touches his nose, repeating the movement several times with the eyes open. This is a more delicate test than the former, both for inco-ordination and intention tremor.

The Trunk. Look for any wasting of the muscles of the trunk. Bend the trunk forward, backwards and to each side and note any limitation of movement.

Ask the patient to sit up without using his hands and note how well he does it.

Lower Limbs. The motor functions are tested as in the upper limbs.

(1) Position of limbs at rest.

(2) Wasting, generalized or local.

(3) Power in all joints—on active movement and resisting movement.

(4) Tone.

(5) Co-ordination Heel-toe test. Toe-finger test. The examiner holds out his finger some distance above the patient's foot. The latter endeavors to touch it with his great toe, raising the foot from the bed. When inco-ordination is present the foot describes wide arcs in the attempt of the toe to reach its objective.

(6) Involuntary movements.

Gait.

This is the most important part of the examination. The movements of the arms must be carefully watched. A hypotonic arm will not be swung as well as the normal arm. A hypertonic arm will likewise fail to move normally; in paralysis agitans the movements of the arms while walking suffer the same fate as many other "associated" movements in this disease and are diminished or absent.

The gaits characteristic of many neurological diseases must be seen and studied, e.g., spastic, ataxic, steppage, scissors, propulsive, etc. The hysterical gait is too grotesque to be mistaken for anything else.

Rhomberg's sign is tested, first with the eyes open and later with them closed.

Reflexes.

The following deep reflexes are tested as a routine: biceps, triceps, supinators, quadriceps and achilles.

The superficial reflexes to be tested in addition to those already mentioned are the plantars, abdominals and cremasterics.

The attempt is made to elicit ankle and patellar clonus. The organic reflexes are investigated: Bladder—is there difficulty in starting the flow, urgency, precipitancy or incontinence? can the patient hold his water too well? Bowels—is there incontinence or severe constipation?

Sensory System

The sensations of light touch, heat and cold, and pain must be tested over the entire body surface These sensations should not all be tested at one examination in detail if sensory loss is present, because patients tire quickly and their answers become unreliable. To insure accuracy attempts are made to trap the patient while testing sensation. Care must be exercised to in no way "suggest" any sensory disturbance to the patient.

If anaesthesia is suspected to be hysterical in origin it can be easily proven. The pin point is moved rapidly over the anaesthetic area towards an area of normal sensation, noting carefully where the change in sensibility occurs (it is usually abrupt in hysteria). Later return to the same place only proceed from the normal area towards the anaesthetic skin. The difference in position of the dividing line on the two examinations is often striking. It is often possible to "suggest" sensitive spots in anaesthetic regions to hysterical patients.

In testing pain sensation with pin prick it is important to always employ the same degree of pressure. This comes with practice and in accurately mapping out areas where sensation is only moderately diminished it is absolutely necessary.

Vibration sense — is often difficult to test and impairment is frequently of great significance in diagnosis It may be tested on all bony prominences on the limbs, hips and spine As with other forms of sensation one should attempt to trap the patient at frequent intervals to be sure he is answering correctly

Deep pressure pain is tested by squeezing hard the muscles of the limbs between the fingers and thumb.

Stereognosis should be tested in the hands as a routine. The compass test for two point discrmiination need only be used in selected cases.

Sense of position and passive movement — is tested in the upper limbs by having the patient close his eyes and hold his arms outstretched in front. He is told to keep the arms in position and not to allow them to move. If sense of position is defective in one arm it will soon move up or down unknown to the patient while the normal will remain stationary. The ability of the patient to detect slight movements at the phalangeal joints is tested, normally very small passive movements of the fingers are appreciated.

The lower limbs are examined in a similar fashion. One limb may be

placed in a certain position (the patient's eyes are shut) and he is told to imitate the position exactly with the other limbs. The big toes are passively flexed and extended, while the patient, with eyes shut, endeavors to indicate the nature of the movement. Normally movements of the toes must be fairly gross to be fully appreciated.

The bones and joints of the skin should be thoroughly examined. The finding of charcot joints, perforating ulcers, herpetic eruptions, neoplasms, etc., are of great aid in diagnosis.

Needless to say all the other systems in the body must be thoroughly examined before a neurological investigation can be considered complete.

I have not mentioned the examination of the cerebro-spinal fluid and several other aids in diagnosis, many of which are of a specialized nature, as they are beyond the scope of this article.

RADIUM AND X-RAY

By A. L. HESSIN, '38.

Little time is devoted to the topic radiology, and the subjects pertinent to it, in the undergraduate years of medicine; no doubt because more basic essential phenomena, theories, and postulations must be dealt with in the time allotted. However, it is a topic which should receive the attention and consideration which is its due in order to realize its place in therapeutics and to create food for thought regarding its possibilities. An explanation of radium, its nature, and its relation to X-ray may serve as an initial step towards the grasping of its as yet unknown potentialities.

Historical Background.—The discovery of Ohm's law in 1825, showing the relation between current and potential difference for solid conductors led to an investigation of the principles underlying the conduction of electricity in liquids and gases. Out of Faraday's work on liquids came the laws of electrolysis and from the work of Crookes and others on gases came a knowledge of cathode rays and Roentgen's discovery of X-rays.

Immediately following Roentgen's discovery in 1895, Bacquerel, in 1896, discovered radioactivity and in 1897 J. J. Thomson showed cathode rays to be particles of matter having a negative charge and mass. In 1898 Madame Currie discovered radium, and in 1900 Rutherford separated the rays from radium into three types called alpha, beta and gamma rays.

Cathode Rays.—An understanding of cathode rays will serve as an introductory step to the phenomenon of X-ray.

A current of electricity of high voltage and low amperage, as can be obtained from an induction coil, can jump a gap between two terminals in one direction, i.e., from the cathode to the anode, and produce a visible spark. If the two terminals are placed within a sealed glass tube and the air gradually withdrawn, the ease with which the jump is made is increased considerably. It follows that by removing air particles, then, whatever is passing from one terminal to the other is not being impeded to such an extent. It is logical enough to assume thereby that the air particles are in the way.

A bullet when fired against a block of steel has its kinetic energy transferred to some new form of energy, by far the greatest quantity of which appears as heat. At least two other forms of energy are produced, i.e., sound and light. So in the case of the entities passing from the cathode to the anode do we have light appearing, as well as other forms, where their kinetic energy is transformed in impact with air particles. Let the anode be moved to one side and a zinc sulphide screen be placed in the path of these entities and

light is given off. It is known as fluorescent light. The streams of entities consist of negatively charged particles and the name cathode rays was given them since they emanated from the cathode. That the particles are negatively charged can be verified by attraction and repulsion of the stream with the positive and negative poles, respectively, of a magnet. J. J. Thomson and others showed beyond doubt that cathode rays were negatively charged particles having a constant negative charge and mass. They were called electrons and are now known to be one of the constituents of all matter.

X-rays.—While investigating the spectrum of the fluorescent light emitted from various materials when bombarded with cathode rays, Roentgen placed a piece of black paper over the cathode ray tube, thereby stopping all visible light. To his surprise he found that certain crystals such as barium platino-cyanide continued to fluoresce when kept in the vicinity of the discharge tube. Having made sure that there were no leaks he decided that this fluorescence was due to some radiation which had the power of penetrating the heavy black paper. He called this new radiation X-rays. These new rays are not corpuscular in nature, nor have they any mass or electrical charge like cathode rays.

It was soon learned that the penetrating qualities of X-rays increased with the speed of the electrons in the discharge tube, which in turn depended on the voltage applied across its electrodes as well as on the gas pressure in the tube. The hardness of the radiation obtained also depended on the material of the anode or the anti-cathode as it is often called.

The nature of X-rays remained a secret for a considerable time, but it gradually became recognized that they were of the nature of light. There remained, however, the difficulty of reflecting, refracting, and diffracting X-rays, all of which was possible with ordinary light. If X-rays were electromagnetic in nature like light, then we should be able to produce interfenence phenomena and hence measure the wave-length of the radiation. No grating could be made fine enough to produce interference, but with the work of Braggs, Laue and others it became known that the planes of atoms in crystals could be used as a natural grating to produce interference fringes.

It is now universally accepted that X-rays constitute merely a small part of the electromagnetic spectrum, ranging from long wave wireless waves at one end to cosmic rays at the other.

There is a very important property of X-rays which should be discussed, namely, the property of ionizing a gas. Ionization is the making of gas a comparatively good conductor of electricity by the formation of electrically charged particles (positive and negative) in the gas which move under the influence of an applied electric field, the positively charged particles moving to the cathode and the negatively charged particles moving to the anode, thus constituting an electric current in the gas.

The discovery of radium.—Shortly after the discovery of X-rays, Becquerel in Paris was investigating the phosphorescence of minerals and he put some unexposed mineral into the cabinet in which he kept his photographic plates. After some days he found his plates to be fogged; he put some more fresh plates in the cabinet and found these fogged in turn. By a process of elimination be found that this effect was due to certain minerals which were known to contain uranium—that is, Pitchblende. Whereupon Madame Curie investigated the mineral and found that the rays were produced by the disintegration of the uranium atom, and that in the process of disintegration a new element was produced. This new element was called radium, and is many times more radioactive than uranium.

Radium rays.—It was soon discovered that the rays from radium were three-fold and they were called alpha, beta and gamma rays. These rays could be readily separated, using magnetic

or electric field methods which forced the alpha rays to one side of a beam, the beta rays to the opposite side and left the gamma rays unaffected. *Alpha rays*: The alpha rays were shown to be positively charged particles moving at relatively slow velocities of about 12,000 to 18,000 miles per second. They were later found to be the nucleus of the helium atom. They can be completely absorbed or stopped by 3 inches of air or a sheet of paper. *Beta rays*: These rays were found to be negatively charged particles, that is, electrons similar to cathode rays and emitted at a velocity of 60,000 to 180,000 miles per second. They were reduced to half by 1 mm. or soft tissue and completely absorbed by 1 mm. of lead or 4 mm. of alumninum. *Gamma rays*: Gamma rays are similar to X-rays except that they are more penetrating. The velocity of propagation of gamma rays is the same as that for X-rays and ordinary light, that is, 186,000 miles per second. They are capable of penetrating 6 to 8 inches of soft tissue and are almost completely absorbed by 6 inches of lead.

Isotopes.*—Based on the theory of matter in which all matter is assumed to consist wholly of positive and negative charges called protons and electrons, the protons forming the nucleus of an atom of matter, the alpha and beta rays were found to come from the nuclei of the atoms of radioactive substances, hence the product of disintegration would have a new arrangement of protons and electrons in its nucleus and therefore would be a new element. In fact Rutherford, in 1902, put forward this idea in his theory of spontaneous disintegration of radioactive matter, thereby starting much speculation on an investigation into the manner in which this arrangement occurred in all elements and their isotopes.

Considering the radioactive changes from Uranium I to its isotope Uranium II, Uranium I has the atomic number 92 and an atomic weight of 238. This means that it has 238 protons and 238—92=146 electrons in its nucleus and there are 92 outer electrons.

Uranium I changes to Ux_2 when it emits an alpha particle from its nucleus. This means a loss of 4 protons and 2 electrons from the nucleus. Therefore Uranium X_1 has an atomic weight of 234, but the atomic number is reduced by two places in the table, that is, to 90.

Uranium X_1 changes to Uranium X_2 by the emission of a beta particle from its nucleus. This change causes the product Ux_2 to have the atomic number 91, but no change occurs in the atomic weight. Uranium X_2 changes to Uranium II by the further emission of a beta particle from its nucleus. This gives U II the same position as U I on the periodic table, but causes no appreciable change in the atomic weight, which remains at 234.

Definition of radioactivity.—Just as when cathode rays are stopped by ponderable matter and give rise to an electromagnetic radiation (X-rays), so we might expect that the sudden eruption of electrons from matter (beta rays) would give rise to a similar electromagnetic radiation. This is found to be the case, for accompanying the beta rays there are usually gamma rays.

Radioactivity may be defined as the spontaneous emission, from the nucleus of a complex atom, of helium nuclei (alpha rays) or single negative electrons (beta rays) and an electromagnetic radiation (gamma rays) with the production of new forms of chemical matter (new elements or isotopes).

Radioactive decay.—The rate of disintegration of a radioactive element is constant and unalterable, the number of atoms of a radioactive element disintegrating in a given time being a constant percentage of the total number of atoms present. For example, Radium decays to half of its value in 1,690 years. All radioactive decay proceeds exponentially; that is, if a substance disintegrates to half in N years, then

*Isotope is a term applied to either of two bodies which are chemically identical yet differ slightly in atomic weight.

only ¼ will remain in 2N years, 1/8 in 3N years, and so on.

There is an exceedingly wide variation in the rates of decay of different radioactive substances, ranging from a half life period of millions of years to fractions of a second.

The product of disintegration of a radioactive element also undergoes disintegration. This is repeated for each element formed so that there is a series of disintegrations which arise from the original radioactive element, each stage of disintegration overlapping the previous stage. There is then a certain amount of each product present and since the number of atoms which disintegrate in a given time is a definite percentage of the total number of atoms present, then the amount of any element present in a series of radioactive products, each in equilibrium with its parent, will be inversely proportional to its rate of decay. That is, if a product decays one thousand times more quickly than its parent the amount of the product present when in equilibrium with its parent will be only 1/1000 that of the parent element, the additional amount which follows in the course of disintegration being equal to the amount which in turn disintegrates to the next lower product in the scale.

Properties of Radium.—Radium itself belongs to the group of alkali earth metals comprising barium, strontium, calcium and magnesium. It is a pure white metal, and like sodium, rapidly changes to the hydroxide in contact with water vapour in the air; hence it is usually prepared in soluble salts such as the carbonate or sulphate. Radium is always found in contact with its parent uranium, and if no loss of radium has occurred throughout the centuries it will be found in the ratio of 1 to 3,400,000 parts of uranium.

Properties of radium emanation.—When radium disintegrates, the first product is radium emanation which is a gas having a molecular weight of 222. It is highly radioactive and emits alpha rays when disintegrating to form radium A, a solid. The half life period of radium emanation is 3.85 days or almost complete disintegration takes place in 30 days.

Radium C, a further product of disintegration, is the stage at which gamma rays are emitted. These are the rays used therapeutically. But Radium C has a short half life period, in other words it decays very rapidly, in fact, so rapidly that it would be impractical to endeavour to extract it from its parent and use it therapeutically. However, Radium C comes into equilibrium with radium emanation in five hours, so that it is a very short time until the gamma rays reach their maximum intensity.

Radium itself or radium emanation could be used as the parent of Radium C. If each were extracted in a relatively pure state, and sealed hermetically in gold or platinum needles, they would supply gamma rays in a constant quantity until disintegration was complete. That is to say, a radium needle would supply gamma rays for 1,690 years; a radium emanation needle would supply gamma rays for approximately 30 days.

A given quantity of radium C in equilibrium with radium emanation is not so expensive as the same quantity of radium C in equilibrium with radium element itself because of the very short life of radium emanation. Therefore, whenever a required treatment can be met with radium emanation as the parent it will prove more economical than using radium, especially if the number of cases being treated does not warrant the purchase of a large quantity of radium. This is the actual practice in the Province of Ontario since the building of the machine for the extraction of radium emanation in the Physics Building at the University of Toronto.

Theoretically, Uranium itself could be used, but it would take centuries for the radium to come into equilibrium with uranium and then in order to have the effect of one gram of radium, about 3½ tons of it would have to be sealed.

Extraction of radium.—The method

of the extraction of radium from ore consists of several processes in which the first aim is to obtain a cencentrate of radium barium sulphate. The method depends on the fact that barium and radium are very similar in their chemical reactions, and if barium be added to a solution containing radium, then any precipitation of barium in its recovery will bring down the radium with it, even if the radium concentration is below the solubility of pure radium sulphate.

The radium barium sulphate is converted into a chloride or bromide and is put into solution. After repeated fractional distillation the product is suciently concentrated for putting up in needles and other therapeutic applicators.

The final product is usually radium bromide or radium sulphate. The bromide is soluble and hence is convenient for use in a radium emanation plant, while the sulphate, which is highly insoluble, is usually used for needles, placques and other therapeutic applicators.

Radiotherapy. — Radium is used therapeutically in a number of different types of applicators, for it so happens that, in cancer, for example, if X-ray or gamma rays from radium are projected on the cells of the tumor they will be destroyed. The effect may be somewhat analogous to the photo-electric effect in which a beam of light of a certain frequency will knock an electron out of matter such as lead. If it were not for the fact that normal tissue is less sensitive to these rays than tumour tissue the use of X-rays and gamma rays would be very hazardous.

The main problem of the radiologist is so to apply the rays that they will most effectively destroy the malignant cells with a minimum of destruction to the normal tissues. It follows therefore, that the deep-seated tumours present the greatest problem in radiotherapy, since the rays must penetrate so much normal tissue in order to reach the growth.

It is believed that the shorter the wavelength of the radiation used, the more selective is its action on the cells; hence for deep therapy work a heavy filtration, not unlike a shield, is used on the applicator, for the purpose of absorbing the longer wavelengths. That is to say, the amount of radiation absorbed by a given filter is greater for the longer wavelengths than for the shorter, hence one hopes to gain selectivity by screening off as much of the softer radiation as possible. The amount of filtration which should be used for a given tumour can be decided upon only by an experienced radiologist.

The various forms of applicators may be classified as follows:

1. Needles containing radium which may be applied interstitially with threads attached for removing, are used. In this case the radium is inserted in the tumour. The needles in most common use contain 1 to 10 mg. of radium, and may be left in the tumour for as long as a week in some cases. A needle containing 2 mg. of radium would be 60 mm. long, with a diameter of 2.25 mm. and a wall thickness of .8 mm., having an epproximate value of $120.00.

2. Radium may be put up in platinum tubes containing up to 100 mg. These tubes are used externally and may be applied in wax moulds to fit any portion of the body.

3. A radium placque is a flat disc containing radium distributed uniformly over its surface. It is particularly suitable for surface work.

4. In the treatment of deep-seated tumours, the use of mass radiation is gaining favour. Mass radiation is the name given to that radiation which would be given by a large quantity of radium. The applicator in this case is called a radium bomb or pack. The average amount of radium used at present in this form of applicator is about 4 gms. The bomb should be so designed that it can be readily adjusted and fixed to any position on the patient. It should provide a flexibility in choice of skin distance, area of port of entry and

filtration. It should, of course, provide ample protection for both the patient and the operator. The Ontario Department of Health is making a thorough investigation of the advantages and disadvantages of this type of applicator in the treatment of cancer of the stomach and intestines.

5. As has been already mentioned, radium emanation may be put up in applicators and used therapeutically in exactly the same way as radium. The gas is usually sealed up in glass capillaries or in small gold tubes called "seeds." These seeds are put in the tumour by means of a trocar and left there. In 30 days the radium emanation will have practically disappeared and the gamma radiation ceased.

It is impossible to deal exhaustively with this topic within the limits of an article of this character. However, having attempted to point out the properties and some of the therapeutic uses of radium, it is hoped that enough has been suggested to prompt further investigation on the part of some readers who, since radiotherapy is not included in the curriculum of medicine, may not previously have had occasion to realize the opportunities which this field has to offer.

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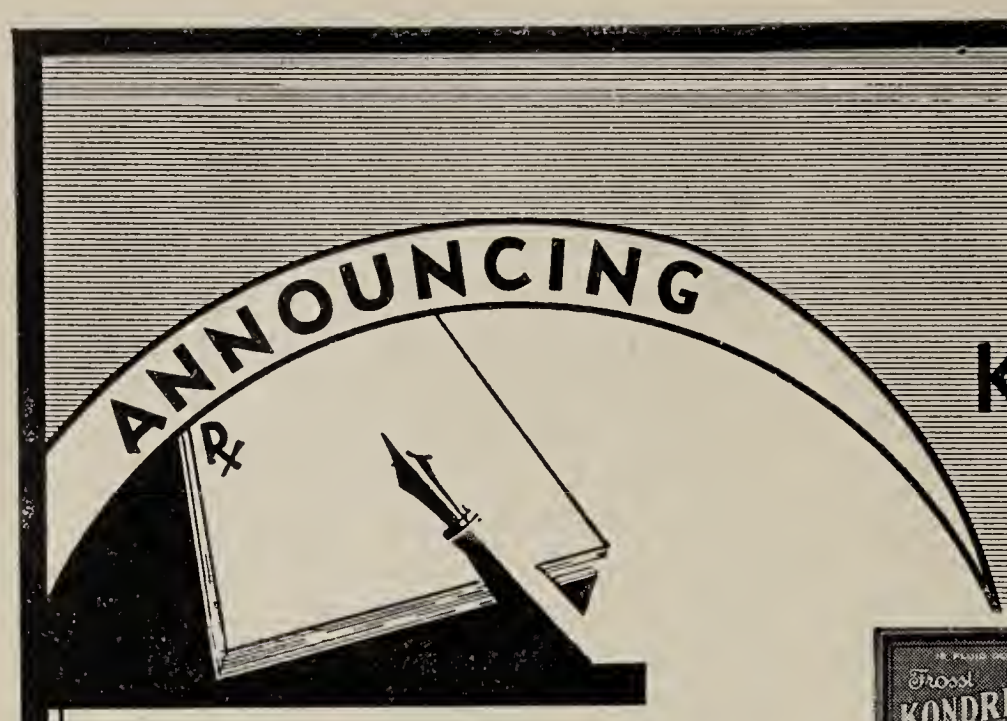

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